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Review Article

Racial and Ethnic Disparities Across the MASLD/MASH Care Continuum in the United States: A Narrative Review

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ARTICLE INFO

Article history:
Received 1 Jan. 2026
Received in revised form 15 Feb. 2026
Accepted 19 Jun. 2026
Published 11 Jul. 2026

Keywords:
MASLD
MASH
Health disparities
Resmetirom
Semaglutide
PNPLA3
Genetic ancestry
Social determinants of health

ABSTRACT

Introduction: Metabolic dysfunction-associated steatotic liver disease (MASLD) affects 25.6% of U.S. adults and is the fastest-growing indication for liver transplantation. The 2023 MASLD nomenclature change, 2024 FDA approval of resmetirom, and 2025 accelerated approval of semaglutide for metabolic dysfunction-associated steatohepatitis (MASH) reshaped the therapeutic landscape. Yet, racial and ethnic disparities persist across the U.S. care continuum.
Methods: This narrative review searched PubMed, MEDLINE, Embase, and Cochrane CENTRAL from inception through 30 April 2026 for English-language studies on racial and ethnic disparities in MASLD epidemiology, screening, fibrosis progression, outcomes, trial representation, and pharmacotherapy access. Guidelines, systematic reviews, observational studies, and randomized trials were prioritized; no PRISMA methodology was used.
Results: Hispanic/Latino adults bear the highest MASLD prevalence (pooled 41%; relative risk vs. non-Hispanic adults 1.50, 95% CI 1.32–1.69), with disproportionate biopsy-cohort MASH (61%), advanced fibrosis (27%), and lower transplant probability (sHR 0.793, 95% CI 0.747–0.842). Non-Hispanic Black adults exhibit lower MASLD prevalence on adjusted NHANES but higher mortality and lower transplant rates in cirrhosis. Noninvasive tools show reduced accuracy in Hispanics (false-negative rates 14%–21%). MAESTRO-NASH enrolled only 2% of Black participants. Resmetirom costs $47,000–$57,670 annually; GLP-1 prescribing disparities in obesity persist across all periods.
Conclusions: Disparities accumulate across the continuum and reflect the interplay among genetic ancestry, social determinants of health, and structural barriers, rather than race or ethnicity as biological variables. An equity framework distinguishing clinical actions, implementation priorities, and research gaps is proposed. No intervention has been demonstrated in MASLD-specific studies to reduce inequities.

1. Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease worldwide, affecting approximately 30% – 40% of the general adult population globally and 60% – 70% of individuals with type 2 diabetes; [1] in the United States, the most recent national NHANES-based estimate of adult MASLD prevalence is lower, at 25.6% (see Epidemiology, below). In 2023, a multisociety Delphi consensus process led by three major international liver associations replaced the terms nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH) with MASLD and metabolic dysfunction-associated steatohepatitis (MASH), respectively [1]. This nomenclature change was motivated by the recognition that the prior terminology relied on exclusionary confounder terms and employed potentially stigmatizing language, while failing to highlight the central pathogenic role of metabolic dysfunction [1]. The new definition requires the presence of hepatic steatosis plus at least one of five cardiometabolic risk factors: overweight/obesity, prediabetes or type 2 diabetes, hypertension, elevated triglycerides, or low HDL cholesterol [1].

Importantly, the 2023 nomenclature framework also introduced the category of MetALD (MASLD with increased alcohol intake) to describe individuals who meet metabolic criteria for MASLD but consume alcohol above thresholds previously used to exclude NAFLD (typically 20 – 50 g/day for women and 30 – 60 g/day for men). The older NAFLD/NASH literature excluded patients with substantial alcohol use, whereas the contemporary MASLD framework explicitly categorizes them. This boundary has practical consequences for disparity research: patients reclassified as MetALD or as alcohol-associated liver disease are not always reported separately in older epidemiologic and registry studies, alcohol use is itself patterned by social and demographic factors (with culturally specific patterns of consumption and reporting), and underreporting is well-documented across all groups. As a result, prevalence estimates, comparisons of fibrosis progression, and transplant outcome data drawn from heterogeneous diagnostic-era cohorts may classify the same individual differently across studies, and apparent racial or ethnic differences may partly reflect differential misclassification rather than true biological differences [1].

The clinical significance of MASLD extends well beyond the liver. Cardiovascular disease is the leading cause of death in individuals

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Citation: Bandaranaike R, Kewish M, Tarwater S. Racial and Ethnic Disparities Across the MASLD/MASH Care Continuum in the United States: A Narrative Review. ASIDE GI. 2026;2(3):32-43, doi:10.71079/ASIDE.GI.071126795

with MASLD, followed by extrahepatic cancers and liver-related complications [1, 2]. However, as fibrosis severity increases, liver-related mortality becomes the predominant cause of death, with five-year mortality reaching 8.3% among patients with cirrhosis [3]. MASH is now the fastest-growing indication for liver transplantation in the United States and the leading cause of liver transplantation among women [4, 5].

The therapeutic landscape for MASH has undergone a historic transformation. In March 2024, resmetirom (Rezdiffra) became the first FDA-approved pharmacotherapy for noncirrhotic MASH with moderate to advanced fibrosis (F2 – F3) [6]. In August 2025, semaglutide (Wegovy) received accelerated FDA approval for the same indication based on the phase 3 ESSENCE trial [7, 8]. These milestones, while representing major advances, also raise urgent questions about equitable access to screening, diagnosis, and treatment across racial and ethnic groups.

This narrative review examines racial and ethnic disparities across the MASLD care continuum, organized into four domains: (1) epidemiology and genetic susceptibility; (2) screening and diagnostic accuracy; (3) fibrosis progression and clinical outcomes; and (4) access to emerging pharmacotherapies and liver transplantation. The review is U.S.-focused: the population data, screening pathways, payer structures, clinical-trial enrollment statistics, and transplant-allocation findings discussed reflect the U.S. health system and U.S. racial/ethnic categorization (the U.S. Office of Management and Budget standard), and findings cannot be assumed to generalize to other countries or health systems where disease burden, ancestry distribution, screening infrastructure, drug pricing, and transplant allocation differ substantially [9]. Where international evidence is cited (for example, the geographically diverse ESSENCE trial), this is noted in the text. An equity-focused framework for addressing U.S. disparities is proposed.

2. Methods

This is a narrative (non-systematic), U.S.-focused review of racial and ethnic disparities across the MASLD care continuum. It was not registered as a systematic review and was not conducted in accordance with PRISMA reporting standards; accordingly, no formal risk-of-bias assessment, evidence-quality grading (e.g., GRADE), or meta-analytic pooling was performed, and evidence selection reflects expert appraisal by the authors rather than predefined eligibility-based screening. PubMed, MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials were searched from each database's inception through the final search date of 30 April 2026. The search combined controlled-vocabulary and free-text terms across three concept blocks: (i) disease terms ("MASLD," "MASH," "NAFLD," "NASH," "metabolic dysfunction-associated steatotic liver disease," "metabolic dysfunction-associated steatohepatitis"); (ii) population and disparity terms ("health disparities," "racial disparities," "ethnic disparities," "Hispanic," "Latino," "Black," "African American," "Asian," "social determinants of health," "health equity"); and (iii) clinical and intervention terms ("PNPLA3," "FIB-4," "transient elastography," "magnetic resonance elastography," "resmetirom," "semaglutide," "GLP-1 receptor agonist," "liver transplantation," "clinical trial representation"). Concept blocks were combined with the Boolean operator AND, and terms within each block with OR. Only English-language publications were eligible. Reference lists of identified articles, recent clinical practice guidelines, and selected review articles were hand-searched to identify additional studies. The initial search returned approximately 1,840 unique records after deduplication; titles and abstracts were screened by the lead author (R.B.) for topical relevance; approximately 220 full-text articles were assessed; and 32 sources were ultimately cited in this review. Because the review synthesizes evidence across several domains, sources were prioritized when they (i) provided the highest available level of evidence for a given claim – in descending order of preference: clinical practice guidelines from the AASLD, AACE, ADA, and major international liver societies; systematic reviews and meta-analyses; randomized controlled trials and large prospective cohort studies; large registry analyses; FDA prescribing information and drug labels; and well-conducted retrospective cohorts and cross-sectional analyses; (ii) reported race- or ethnicity-stratified results; (iii) used contemporary MASLD/MASH diagnostic criteria where available; or (iv) were published since 2020 and addressed therapies, screening tools, or policy changes most relevant to current practice. Case reports, conference abstracts not subsequently published as full articles, and editorials were excluded unless they provided unique data not available elsewhere. The authors performed selection without independent duplicate screening; readers should therefore interpret this review as an evidence-informed narrative synthesis rather than a comprehensive systematic appraisal. The complete final search strategy for each database – including controlled-vocabulary and free-text terms, database-specific syntax, date limits, and language filters is provided as Supplementary Material (Appendix S1).

Throughout this review, we use the terms "race" and "ethnicity" as social rather than biological constructs, consistent with current guidance from the National Institutes of Health and the U.S. Office of Management and Budget; these are self-identified categories that capture social experience and structural exposure rather than genetic differences. When we discuss genetic data such as PNPLA3 allele frequency, we use the term "genetic ancestry" to refer to genome-derived population-of-origin information, which is correlated with, but not equivalent to, self-identified race or ethnicity. Where individual studies reported demographic categories differently from this convention, we have preserved each study's original terminology. For every effect estimate discussed, we have endeavored to specify whether it is unadjusted, age- and sex-adjusted, or fully multivariable-adjusted; where the original study did not make this clear, we describe the estimate as "reported" rather than "adjusted." Many of the cited estimates derive from observational designs in which residual confounding by age, sex, diabetes, obesity, insurance status, socioeconomic position, geography, and care-setting factors cannot be fully excluded, and we have tempered the corresponding interpretations accordingly.

3. Results

3.1. Epidemiology: Differential Burden by Race and Ethnicity

3.1.1. Prevalence Disparities

The prevalence of MASLD varies substantially by race and ethnicity in the United States. Using transient elastography data from the National Health and Nutrition Examination Survey (NHANES) 2017 – 2023, the overall prevalence of MASLD was estimated at 25.6%, with a prevalence of fibrosis at 11.3% [10]. Notably, between the two survey cycles, age-standardized MASLD prevalence decreased (26.8% to 23.6%), while fibrosis prevalence increased (10.4% to 12.7%), suggesting a shift toward more advanced disease in the population [10]. A summary of prevalence and fibrosis rates by race/ethnicity is presented in (**Table 1**).

Hispanic/Latino individuals bear the highest burden. A systematic review and meta-analysis of 22 studies comprising 756,088 subjects found a pooled MASLD prevalence of 41% among U.S. Hispanic adults, with 61% prevalence of MASH and 27% prevalence of

Table 1: MASLD Burden by Race/Ethnicity in the United States

Comparison Groups	<65 vs >75 (mean rank difference, p value)	<65 vs 65–75 (mean rank difference, p value)	65–75 vs >75 (mean rank difference, p value)	All age groups (H(df), p value)
Racial/Ethnic Group	Estimate (with reported 95% CI or range where available)	Source Population / Denominator	Diagnostic Method	Key Reference
Panel A. Population-Level MASLD Prevalence (general adult population)				
Overall U.S.	MASLD 25.6%; fibrosis 11.3%; age-standardized prevalence trend 26.8% → 23.6% across survey cycles	U.S. adults, NHANES 2017–2023 (n ≈ 13,500)	VCTE-based CAP and LSM	Unalp-Arida & Ruhl, 202510
Hispanic/Latino	MASLD 41% (pooled point estimate). Multivariable-adjusted RR vs. non-Hispanic adults: 1.50 (95% CI 1.32–1.69) for MASLD; 1.42 (95% CI 1.04–1.93) for MASH.	U.S. Hispanic adults, meta-analysis of 22 studies (n = 756,088)	Mixed (imaging, biochemistry, biopsy across constituent studies)	Tesfai et al., 202511
Non-Hispanic Black	Lower than non-Hispanic White (inverse association on multivariable-adjusted analysis)	U.S. adults, NHANES 2017–2023	VCTE-based CAP and LSM	Unalp-Arida & Ruhl, 202510
Non-Hispanic Asian	Elevated (significant positive association on multivariable-adjusted analysis)	U.S. adults, NHANES 2017–2023	VCTE-based CAP and LSM	Unalp-Arida & Ruhl, 202510
Panel B. Disease Severity Within Biopsy-Proven MASLD Cohorts (already-diagnosed patients only; NOT a population-level estimate)				
Hispanic/Latino	MASH 61%; advanced fibrosis (≥F3) 27% (pooled point estimates within biopsied subset)	Hispanic adults with biopsy-proven MASLD, meta-analysis subset	Liver biopsy	Tesfai et al., 202511
Non-Hispanic White	Definite MASH 58%; advanced fibrosis (≥F3) 34%; cirrhosis 12.1%. Reference group for Samala adjusted OR comparison.	Biopsy-proven NAFLD; NASH CRN (n = 1,910 NHW of 2,019 total)	Liver biopsy	Samala et al., 202413
Non-Hispanic Black	Definite MASH 59%; advanced fibrosis (≥F3) 22%; cirrhosis 4.6%. Multivariable-adjusted OR for advanced fibrosis vs. NHW: 0.48 (95% CI 0.27–0.86), adjusted for clinical risk factors and PNPLA3 genotype.	Biopsy-proven NAFLD; NASH CRN (n = 109 NHB of 2,019 total)	Liver biopsy	Samala et al., 202413
Panel C. PNPLA3 rs738409 G Allele Frequency by Genetic Ancestry (not a disease prevalence estimate; values are population ranges across reference samples)				
Hispanic/Latino (high Amerindian ancestry)	47%–52% (range across reference samples)	Genetic reference populations	Genotyping	Cumpian et al., 202512
Non-Hispanic White	15%–23% (range across reference samples)	Genetic reference populations	Genotyping	Cumpian et al., 202512
Non-Hispanic Black	14%–17% (range across reference samples)	Genetic reference populations	Genotyping	Cumpian et al., 202512
Non-Hispanic Asian (subgroup-heterogeneous)	Variable (heterogeneous across East/South/Southeast Asian subgroups)	Genetic reference populations	Genotyping	Cumpian et al., 202512

Advanced fibrosis is defined as bridging fibrosis (stage F3) on the NASH CRN scale. Panel A presents prevalence estimates in the general adult population and is NOT directly comparable to Panel B, which describes severity within already-diagnosed biopsy cohorts. Panel C reports allele frequencies in genetic-ancestry-related reference populations and is NOT a disease-prevalence estimate. Where the source publications reported 95% confidence intervals, the most informative effect estimates (multivariable-adjusted relative risks for Hispanic adults, and the adjusted odds ratio for advanced fibrosis in non-Hispanic Black vs. non-Hispanic White biopsy cohort participants) have been added to the Estimate column.
Abbreviations: AA, African American; CAP, controlled attenuation parameter; CI, confidence interval; LSM, liver stiffness measurement; MASH, metabolic dysfunction-associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease; NAFLD, nonalcoholic fatty liver disease; NASH CRN, NASH Clinical Research Network; NHANES, National Health and Nutrition Examination Survey; NHB, non-Hispanic Black; NHW, non-Hispanic White; OR, odds ratio; PNPLA3, patatin-like phospholipase domain-containing 3; RR, relative risk; VCTE, vibration-controlled transient elastography.

MASH-associated advanced fibrosis (defined as bridging fibrosis, stage F3 on the NASH Clinical Research Network fibrosis scale; this definition is used throughout the present review unless otherwise specified) [11]. Compared with non-Hispanic adults, Hispanic adults had a relative risk of 1.50 (95% CI 1.32 – 1.69) for MASLD and 1.42 (95% CI 1.04 – 1.93) for MASH; these estimates were from multivariable-adjusted models in the constituent studies, but adjustment sets were heterogeneous across the meta-analyzed cohorts [11]. The annual percentage increase in MASLD prevalence among Mexican American and other Hispanic/Latino persons was 1.9% and

Table 2: Racial and Ethnic Representation in Pivotal MASH Clinical Trials

Trial / Source	Drug	Phase	Total N (basis for %)	RACE — % White / % Black or AA / % Asian / % Other	ETHNICITY — % Hispanic/Latino	How reported	% Not reported	Source
MAESTRO-NASH	Resmetirom	3	966 (ITT)	89 / 2 / 3 / ~6	21	Race and Hispanic/Latino ethnicity reported separately per OMB categories	Not specified	Harrison et al., 20246
ESSENCE (interim)	Semaglutide 2.4 mg	3	800 (first interim analysis)	67.5 / NR separately / NR separately / NR separately	NR separately	Race reported only for White; geographic region reported (Asia 25.1%, Europe 25.3%, North America 35.0%, South America 7.9%)	Not specified	Sanyal et al., 20258
Pooled MASH RCTs	Various	1–3	19,516 across 112 RCTs (race reported in 61.6% of trials; ethnicity in 50.0%)	87.6 / 2.3 / 5.0 / ~5	31.4	Pooled proportions; denominators differ between race and ethnicity because not all trials reported both	38.4 (race); 50.0 (ethnicity)	Souza et al., 202532

Per OMB categorization, Hispanic/Latino ethnicity is independent of race; the same individual may be reported under both a race category and the Hispanic/Latino ethnicity category. Percentages within a single column may therefore exceed 100% across rows of a single study, and Hispanic/Latino percentages are not subtractive from race percentages. No MASLD-specific population denominator is presented in this table; the racial and ethnic enrollment percentages reported in the trials above are therefore not directly comparable with the racial/ethnic composition of the U.S. MASLD-affected population, and no such comparison should be inferred.

Abbreviations: AA, African American; ITT, intention to treat; MASH, metabolic dysfunction-associated steatohepatitis; NR, not reported; OMB, U.S. Office of Management and Budget; RCT, randomized controlled trial.

1.7%, respectively, compared with 1.1% in non-Hispanic Black and non-Hispanic White populations [12].

In contrast, non-Hispanic Black individuals consistently demonstrate lower MASLD prevalence. In multivariable-adjusted NHANES analyses (adjusted for age, sex, BMI, diabetes, hypertension, dyslipidemia, and insurance status), both MASLD and fibrosis were inversely associated with non-Hispanic Black race/ethnicity [10]. This finding persists after controlling for metabolic risk factors and the PNPLA3 rs738409 polymorphism in biopsy cohorts, representing one of the most striking and incompletely understood paradoxes in MASLD epidemiology [13]. As discussed below, the apparent paradox should be interpreted with attention to ascertainment differences and to the predominantly tertiary-care setting from which the supporting biopsy cohort is drawn.

Non-Hispanic Asian individuals also demonstrate elevated MASLD risk, with NHANES data showing a significant positive association between non-Hispanic Asian race/ethnicity and MASLD on multivariable adjustment [10]. This population is particularly notable for developing MASLD at lower body mass index thresholds, a phenomenon termed "lean MASLD."

3.1.2. Heterogeneity Within Broad Racial and Ethnic Categories

The labels "Hispanic/Latino" and "Asian" each subsume highly heterogeneous populations whose MASLD risk, PNPLA3 ancestry-allele frequency, immigration history, body composition, dietary patterns, language access, and engagement with the U.S. health system differ substantially. Within U.S. Hispanic/Latino populations, available data suggest the highest MASLD prevalence among Mexican American and Central/South American adults of high Amerindian ancestry, with progressively lower prevalence in adults of predominantly European-Iberian or Afro-Caribbean ancestry; PNPLA3 G allele frequency similarly varies by subgroup, broadly tracking the proportion of indigenous American ancestry [12]. Within U.S. Asian populations, MASLD risk and metabolic phenotype differ between East Asian (Chinese, Korean, Japanese), South Asian (Indian, Pakistani, Bangladeshi, Nepali, Sri Lankan), and Southeast Asian (Filipino, Vietnamese, Thai, Cambodian, Indonesian) subgroups, with South Asians displaying the highest reported prevalence of insulin resistance, abdominal adiposity at low BMI, and MASLD; published U.S. cohorts have rarely been powered to compare these subgroups directly. We discuss findings at the broader "Hispanic/Latino" and "Asian" level only because the underlying studies aggregate at this level; where subgroup-specific data are available, we say so, and where they are not (most of the time), the resulting estimates should be regarded as group-level averages that may obscure clinically meaningful subgroup differences.

3.1.3. Genetic Susceptibility, Genetic Ancestry, and the PNPLA3 Variant

Before discussing PNPLA3, it is important to distinguish three constructs that are sometimes conflated in disparity research. Self-identified race and ethnicity are social categories that capture lived social experience and structural exposure (housing, education, insurance, neighborhood resources, discrimination, language access). Genetic ancestry is a genome-derived measure of population of origin that is correlated with but not equivalent to self-identified race or ethnicity. Social determinants of health are the upstream non-medical drivers (income, education, food security, housing, neighborhood exposures, discrimination) that operate independently of either. PNPLA3 allele frequency is most properly interpreted as a marker of genetic ancestry; its higher frequency in U.S. Hispanic/Latino populations of high Amerindian ancestry helps explain part of the disease-prevalence gradient, but it does not explain (and should not be invoked to explain) structural disparities in screening, diagnosis, treatment, or transplant access. Framing population-level differences as if they reflected race or ethnicity as biological variables is scientifically inaccurate and risks reinforcing biologistic-determinist interpretations of predominantly socially structured outcomes.

Table 3: Disparities in Liver Transplantation — Labeled by Comparison Type

Outcome	Comparison	Effect Estimate	95% CI	p Value	Key Reference
Section A. Race/ethnicity-specific comparisons within NASH/MASH cirrhosis (UNOS registry, n = 18,562; adjusted for age, sex, MELD, blood group, transplant region, donor service area)					
Transplant probability	Hispanic vs. non-Hispanic White	sHR 0.793	0.747–0.842	<0.001	Lim et al., 202326
Waitlist mortality	Hispanic vs. non-Hispanic White	sHR 1.173	1.052–1.308	0.004	Lim et al., 202326
MELD score at listing	Black vs. non-Hispanic White	Effect size 2.307	1.561–3.053	<0.001	Lim et al., 202326
Section B. Race-specific comparisons in cirrhosis of ALL etiologies (NOT MASH-specific; adjusted for age, sex, MELD, insurance, neighborhood deprivation, primary cirrhosis etiology); included for context only					
Liver-related death	Black vs. non-Hispanic White (all-cause cirrhosis, n = 11,277)	sHR 1.26	1.15–1.38	<0.001	Mazumder et al., 202125
Transplant listing rate	Black vs. non-Hispanic White (all-cause cirrhosis)	Lower	—	<0.001	Mazumder et al., 202125
Section C. Sex × race/ethnicity interaction within NASH cirrhosis (UNOS; women included)					
MELD score at transplant	Non-White women vs. White women	26.1 vs. 23.1	—	<0.001	Becker et al., 202427
Post-transplant graft survival	Non-White women vs. White women	Worse	—	<0.05	Becker et al., 202427
Post-transplant patient survival	Non-White women vs. White women	Worse	—	<0.05	Becker et al., 202427
Section D. Disease-level comparison (NASH cirrhosis vs. non-NASH cirrhosis); not race-stratified; included to show structural disadvantage of MASH within the allocation system					
Transplant probability at 90 days	NASH vs. non-NASH cirrhosis	HR 0.873	—	<0.001	Lim et al., 20235
Transplant probability at 1 year	NASH vs. non-NASH cirrhosis	HR 0.867	—	<0.001	Lim et al., 20235

Sections A and D data are from the UNOS registry analysis of 18,562 patients with NASH cirrhosis listed between 2000 and 2021. Section B data are from a metropolitan cohort of 11,277 patients with cirrhosis of all etiologies and are included for context; they should not be interpreted as MASH-specific outcome data. Section C data are from a UNOS analysis of women with NASH cirrhosis. "—" indicates confidence interval not reported in the original study or not applicable.

Abbreviations: CI, confidence interval; HR, hazard ratio; MELD, Model for End-Stage Liver Disease; NASH, nonalcoholic steatohepatitis; NHW, non-Hispanic White; sHR, subdistribution hazard ratio; UNOS, United Network for Organ Sharing.

With these definitions in place, genetic variation does play a real role in MASLD susceptibility and severity. Heritability accounts for approximately 50% of the variability in MASLD [2]. The most robustly validated genetic determinant is the rs738409 (I148M) variant in the patatin-like phospholipase domain-containing 3 gene (PNPLA3), which impairs lipid-droplet remodeling and promotes hepatic fat accumulation, inflammation, and fibrogenesis [2, 14].

The PNPLA3 G allele frequency, viewed as an ancestry-related variable, ranges across reference populations from approximately 47% – 52% in U.S. Hispanic/Latino samples to 15% – 23% in non-Hispanic White samples and 14% – 17% in non-Hispanic Black samples [12]. The G allele confers an approximately 2.3-fold greater risk of developing MASLD per allele. It is the only common variant robustly and independently associated with histologic MASLD severity in large case-only biopsy cohorts [12, 14]. The higher frequency of the G allele in populations of high South/Central American Amerindian ancestry contributes to, but does not by itself account for, the higher MASLD burden observed in Hispanic/Latino communities; nutrition, food insecurity, occupational exposures, healthcare access, and structural barriers operate alongside genetic ancestry [12].

Importantly, the phenotypic expression of genetic risk factors is modulated by environmental exposures, including overall adiposity, insulin resistance, diet, and alcohol consumption [2]. Patients with PNPLA3 variants appear to require fewer metabolic risk factors before progressing to MASH, suggesting a gene-environment interaction that may be particularly relevant in populations with high ancestry-related allele frequency [12]. Routine genetic screening is not currently recommended; the ancestry-related allele frequencies of risk variants may become a consideration for risk stratification in the future, but should be used at the individual level, based on genotype rather than presumed group membership [12].

Other variants, including TM6SF2 and MBOAT7, also contribute to MASLD susceptibility. However, their effect sizes are smaller, and their associations with histologic severity are less robust than those of PNPLA3 [4, 14]. A multiancestry evaluation of "tier 1" genetic variants across patients with biopsy-characterized MASLD found that variants in the PNPLA3-SAMM50-PARVB locus showed the most consistent associations with fibrosis severity and NAFLD activity score [14].

3.2. Social Determinants of Health

Beyond genetic ancestry, social determinants of health (SDOH) profoundly influence MASLD prevalence and outcomes. Food insecurity – the limited or uncertain access to nutritionally adequate foods – is independently associated with higher odds of developing MASLD and MASLD-associated advanced fibrosis, even after adjusting for poverty status, education level, race, and ethnicity [4, 15]. A dose-dependent relationship exists between unfavorable SDOH composite scores and all-cause mortality among individuals with MASLD: each one-point increase in SDOH score is associated with a 32% increase in hazard for all-cause mortality (HR 1.32, 95% CI 1.25 – 1.38) on multivariable adjustment [16].

Among Hispanic/Latino populations, food insecurity, limited access to health care, language barriers, low health literacy, and uncertain immigration-related health-system engagement compound metabolic and ancestry-related risk to accelerate MASLD progression [12]. Higher educational attainment was significantly associated with lower odds of MASLD among participants with food security (adjusted OR 0.71, 95% CI 0.55 – 0.91) but not among those with food insecurity, suggesting that food security may be a prerequisite for the protective effects of education [17].

3.3. Screening and Diagnostic Disparities

3.3.1. Current Screening Recommendations

Multiple professional societies now recommend screening for MASLD-related fibrosis in high-risk populations. The American Diabetes Association (ADA) 2025 consensus report and the 2026 Standards of Care recommend the Fibrosis-4 Index (FIB-4) as the most cost-effective initial screening strategy for people with prediabetes and cardiometabolic risk factors or with type 2 diabetes [4, 18]. The AASLD and the AACE similarly endorse a staged approach using FIB-4 as a first-line tool, followed by imaging-based assessments such as vibration-controlled transient elastography (VCTE) or magnetic resonance elastography (MRE) for those with indeterminate or elevated scores [19–21].

Despite these recommendations, clinicians consistently underestimate the prevalence of at-risk MASH and do not implement appropriate screening strategies, resulting in sparse referral to specialists and inadequate prescription of medications with potential efficacy [18]. This pattern of underdiagnosis is compounded by the fact that at-risk MASH with clinically significant fibrosis (≥F2) is frequently observed with plasma aminotransferases below the commonly used cutoff of 40 U/L [18].

3.3.2. Differential Performance of Noninvasive Tests

A critical and underappreciated disparity lies in the differential performance of noninvasive screening tools across racial and ethnic groups. In a single prospective study of 244 adults with biopsy-proven MASLD, MRE and VCTE exhibited significantly lower accuracy for detecting significant fibrosis in Hispanic versus non-Hispanic participants (AUROC: MRE, 0.87 vs. 0.98, $p = 0.01$; VCTE, 0.78 vs. 0.92, $p = 0.02$) [22]. Clinical care algorithms yielded high false-negative rates among Hispanic participants in that cohort: 14% with low-risk FIB-4 and 21% with low-risk VCTE had advanced fibrosis on biopsy [22].

FIB-4 is not a race-neutral biological measurement: it is a composite of age, AST, ALT, and platelet count, and each of these components varies systematically by population. The age term contributes substantially to the FIB-4 value and, with standard cutoffs (<1.3 to rule out, ≥2.67 to refer for further assessment), tends to underclassify younger adults and over-classify older adults regardless of underlying liver disease; this may particularly affect younger Hispanic/Latino patients with high genetic-ancestry risk who can have clinically significant fibrosis at younger ages. Coexistent type 2 diabetes affects aminotransferase distributions and modifies the relationship of FIB-4 to histologic fibrosis. Platelet count varies by population, comorbidity, and certain medications (including some antihypertensives and antiretrovirals). AST and ALT distributions are influenced by sex, body composition, muscle mass, recent exercise, and alcohol intake. Population-level shifts in any of these components can change the operating characteristics of FIB-4 in ways that are not captured by race/ethnicity stratification alone. Implementation pathways that propose race- or ethnicity-specific FIB-4 thresholds should therefore be paired with age- and comorbidity-specific calibration; thresholds that vary by self-identified group risk imply biologic difference where age and comorbidity structure is the real driver.

These findings have direct clinical implications. Standard noninvasive test cutpoints were derived from predominantly non-Hispanic populations and may be insufficiently sensitive for Hispanic individuals, who carry the highest disease burden [22]. In the derivation cohort, optimized cutpoints of 2.73 kPa for MRE and 6.9 kPa for VCTE were identified for detecting significant fibrosis in Hispanic populations, and these findings were externally evaluated in a Latin American cohort [22]. A proposed pathway incorporating adjusted FIB-4 and secondary assessment thresholds for Hispanic/Latino patients has been outlined, but these population-specific thresholds should be regarded as investigational. Broader prospective validation in diverse, community-based U.S. populations is required before they are adopted into routine clinical practice, and current professional society guidelines (AASLD, AACE, ADA) do not yet endorse race- or ethnicity-specific cutpoints [12].

Lowering screening thresholds – whether universally or for specified subgroups – has consequences beyond improved sensitivity. A more permissive FIB-4 cutoff increases the number of patients referred for second-line VCTE or MRE assessment; in U.S. settings where VCTE and especially MRE capacity is concentrated in tertiary centers, this translates directly into longer wait times, higher per-encounter cost, and (paradoxically) potentially less access for the very populations the change is meant to serve. The same change increases the number of false-positive results that prompt specialist referral, additional imaging, possible biopsy, and patient anxiety – a non-trivial harm that must be weighed against the gain in true positives. Hepatology capacity in safety-net systems is already strained. An equity-focused screening strategy must therefore be implementable, not only clinically valid; we suggest that any move toward population-specific thresholds be accompanied by health-system modeling of the projected change in referral volume, by investment in community-based VCTE infrastructure, and by primary-care decision-support tools that distinguish high-yield from low-yield referrals.

3.4. Barriers to Screening Implementation

Structural barriers to screening disproportionately affect racial and ethnic minorities. These include limited access to primary care, lack of insurance coverage, language barriers, and low health literacy [12, 23]. The requirement for specialized imaging (VCTE or MRE) as a second-line assessment creates additional access barriers, as these technologies are concentrated in academic medical centers and may not be available in community settings serving predominantly minority populations [24]. Systematic screening for social barriers in hepatology practices has been proposed as a strategy to identify gaps in the care cascade [23].

3.5. Fibrosis Progression and Clinical Outcomes

3.5.1. The Non-Hispanic Black "Paradox" in Context

One of the most-cited findings in MASLD epidemiology is the paradoxically lower prevalence of MASLD and advanced fibrosis in non-Hispanic Black individuals, despite a higher burden of metabolic risk factors in some studies. In a NASH Clinical Research Network biopsy cohort of 109 non-Hispanic Black and 1,910 non-Hispanic White adults with biopsy-proven NAFLD, non-Hispanic Black individuals had similar NAFLD activity scores (4.4 vs. 4.3) and proportions with definite MASH (59% vs. 58%) but significantly lower rates of advanced fibrosis (22% vs. 34%, $p = 0.01$) and cirrhosis (4.6% vs. 12.1%, $p = 0.01$) [13]. After multivariable adjustment for clinical risk factors and PNPLA3 genotype, non-Hispanic Black race remained independently associated with lower odds of advanced fibrosis (adjusted OR 0.48, 95% CI 0.27 – 0.86) [13].

However, among those non-Hispanic Black individuals who did develop advanced fibrosis, the NAFLD activity score (5.6 vs. 4.9, $p = 0.01$) and lobular inflammation grade (2.2 vs. 1.7, $p = 0.002$) were significantly higher than in non-Hispanic White counterparts with advanced fibrosis [13]. This pattern has been interpreted as evidence of a fundamentally different relationship between necroinflammation and fibrogenesis. We urge caution with that inference: the cohort is small ($n = 109$ non-Hispanic Black participants), drawn from tertiary-referral biopsy practices, and subject to substantial ascertainment bias (see Selection, Ascertainment, and Referral Bias, below). The pattern may equally well reflect differential referral for biopsy, differential delay from disease onset to evaluation, or unmeasured confounding.

Despite lower MASLD prevalence, non-Hispanic Black patients with cirrhosis – in studies of all etiologies – face worse outcomes. In a large metropolitan cohort study of 11,277 patients with cirrhosis from all causes (not restricted to MASH cirrhosis), Black patients had the highest rate of all-cause mortality and non-liver-related death and were less likely to be listed or transplanted ($p < 0.001$ for all) [25]. In multivariable competing-risk analysis (adjusted for age, sex, MELD score, insurance, neighborhood deprivation, and primary cirrhosis etiology), Black patients had a 26% increased hazard of liver-related death (sHR 1.26, 95% CI 1.15 – 1.38) [25]. Because that cohort included patients with cirrhosis from viral hepatitis, alcohol-associated liver disease, and other causes in addition to MASH, these findings should not be interpreted as MASLD/MASH-specific outcome data. MASH-specific outcome data in non-Hispanic Black patients remain limited, and the contribution of disparities in care access, comorbidity burden, and competing causes of death to the worse cirrhosis outcomes observed in this population cannot be disentangled from any MASLD-specific biology in the existing literature.

3.6. Hispanic/Latino Populations: From High Prevalence to Adverse Outcomes

Hispanic/Latino individuals face a compounding trajectory of risk: the highest MASLD prevalence, the fastest rate of prevalence increase, and disproportionately adverse outcomes. Overall mortality among Hispanic/Latino patients with MASLD surpasses most other U.S. populations, with significantly increased risk of hepatocellular carcinoma and cirrhosis compared with non-Hispanic/Latino patients in age- and sex-adjusted analyses; the extent to which insurance, neighborhood, and care-access factors confound these comparisons is incompletely characterized [12].

The Global MASLD (G-MASLD) study, enrolling 17,792 patients with biopsy-confirmed MASLD, confirmed that advanced fibrosis (≥F3) was present in 35% of the cohort, with type 2 diabetes prevalence increasing stepwise from 28% in F0 to 70% in F4 [3]. Independent predictors of advanced fibrosis in multivariable models included older age, type 2 diabetes, and obesity; fibrosis severity – whether defined histologically or by noninvasive tests – was strongly associated with higher risks of death and liver-related outcomes [3].

3.7. Liver Transplant Disparities

Disparities in liver transplantation for MASH cirrhosis are well documented and summarized in **(Table 3)**. In an analysis of 18,562 patients with NASH cirrhosis listed in the United Network for Organ Sharing (UNOS) registry (2000 – 2021), Hispanic patients had a lower probability of receiving liver transplantation (sHR 0.793, 95% CI 0.747 – 0.842) and increased waitlist mortality (sHR 1.173, 95% CI 1.052 – 1.308) compared with non-Hispanic White patients on multivariable adjustment for age, sex, MELD score, blood group, transplant region, and donor service area [26]. African American patients were listed with significantly higher Model for End-Stage Liver Disease (MELD) scores (effect size 2.307, $p < 0.001$) than non-Hispanic White patients [26].

Additional disparities exist at the intersection of race/ethnicity and sex. Non-White women with NASH cirrhosis were transplanted at higher MELD scores (26.1 vs. 23.1, $p < 0.001$) than White women and had worse graft and patient survival after transplantation [27]. These data indicate that non-White women with MASH represent the most vulnerable population on the U.S. liver transplant waitlist [27].

Patients with NASH cirrhosis as a whole face structural disadvantages in the transplant allocation system. Compared with non-NASH cirrhosis, NASH cirrhosis is associated with lower transplant probability at 90 days (HR 0.873, $p < 0.001$) and 1 year (HR 0.867, $p < 0.001$), and higher waitlist mortality [5]. Serum creatinine, rather than bilirubin, is the major contributor to MELD score increases leading to transplantation in NASH cirrhosis, suggesting that the MELD score may not optimally capture mortality risk in this population [5].

3.8. Access to Emerging Pharmacotherapies

3.8.1. Resmetirom (Rezdiffra)

Resmetirom, a liver-directed thyroid hormone receptor beta (THR-β) selective agonist, received FDA accelerated approval in March 2024 based on the MAESTRO-NASH trial [6]. In this phase 3 trial, 966 patients with biopsy-proven MASH and F1 – F3 fibrosis were randomized to resmetirom 80 mg, 100 mg, or placebo for 52 weeks. MASH resolution without worsening of fibrosis was achieved in 25.9% (80 mg) and 29.9% (100 mg) versus 9.7% (placebo), while fibrosis improvement without worsening of MASH was achieved in 24.2% (80 mg) and 25.9% (100 mg) versus 14.2% (placebo) ($p < 0.001$ for all comparisons) [6].

The trial population was demographically homogeneous and underrepresented racial and ethnic minorities relative to the U.S. MASLD-affected population: 89% White, 21% Hispanic, 3% Asian, and only 2% Black or African American **(Table 2)**[28]. This severe underrepresentation of Black patients limits the generalizability of efficacy and safety data to this population. Current practice guidance on resmetirom use in noncirrhotic MASH does not address race- or ethnicity-specific considerations for patient selection or monitoring.

The cost of resmetirom presents a significant barrier to equitable access. The average annual wholesale price is approximately $47,000 – $57,670, and the medication may not be covered by health insurance or may require prior authorization [29]. An economic evaluation found that at a price of $19,011/year, resmetirom was not cost-effective compared with standard of care at a

$100,000/QALY willingness-to-pay threshold [29]. At the current wholesale price, the incremental cost-effectiveness ratio was estimated at $140,134/QALY [29]. In comparison, off-label treatments such as pioglitazone are available generically at a small fraction of the cost of FDA-approved MASH therapies.

3.8.2. Semaglutide (Wegovy)

Semaglutide received accelerated FDA approval in August 2025 for the treatment of noncirrhotic MASH with moderate to advanced fibrosis (F2 – F3) based on the phase 3 ESSENCE trial [7]. In the first 800 participants, 72 weeks of semaglutide 2.4 mg/week achieved resolution of MASH without worsening of fibrosis in 62.9% versus 34.3% with placebo ($p < 0.001$), and improvement in fibrosis without worsening of MASH in 36.8% versus 22.4% ($p < 0.001$) [7, 8].

The ESSENCE trial enrolled a geographically more diverse population than MAESTRO-NASH, with participants from Asia (25.1%), Europe (25.3%), North America (35.0%), and South America (7.9%) (Table 2) [8]. However, the trial investigators themselves acknowledged limitations including the small number of Black patients and a lack of data regarding genetic polymorphisms as determinants of therapeutic response [8]. The trial population was 67.5% White and 57.1% female [8].

3.8.3. The Real-World MASH Treatment-Access Pathway: Where Patients Are Lost

Issuing an FDA-approved MASH therapy is not the same as a patient actually receiving it. The pre-prescription pathway in the United States involves several sequential steps, each of which is associated with documented or plausible disparities. (1) Risk identification in primary care depends on systematic application of cardiometabolic criteria; underdiagnosis is well-documented and is greater in patients with low health literacy, limited English proficiency, and intermittent care engagement. (2) Fibrosis assessment with FIB-4 is inexpensive but is not routinely ordered; second-line VCTE or MRE requires referral and equipment that is unevenly distributed (see Barriers to Screening Implementation). (3) Specialist referral to hepatology or to a metabolic-MASH program adds a wait of weeks to months and depends on insurance network, transportation, and local capacity. (4) Confirmation of eligibility requires documentation of noncirrhotic F2 – F3 fibrosis and exclusion of cirrhosis; some payers also require demonstration of failed lifestyle intervention. (5) Prior authorization is universal for resmetirom and is increasingly common for GLP-1 receptor agonists [30]; approval rates vary by payer and have been shown in obesity prescribing data to be lower for patients in higher-deprivation neighborhoods [31]. (6) Treatment initiation requires that the patient can afford copays (typically $20 – $400/month with insurance, full wholesale cost without it) and can manage the route of administration (oral resmetirom vs. injectable semaglutide). (7) Monitoring and follow-up (addressed below) introduces further demands. Loss at any of these steps disproportionately affects patients with limited insurance, limited English, rural residence, or precarious work schedules. Disparities in actual MASH-pharmacotherapy use will therefore exceed disparities at any single step, and reporting only "prescribed/not prescribed" undercounts the problem.

3.8.4. Safety Monitoring and Tolerability as Equity Issues

The cost discussion has tended to dominate the equity literature on new MASH therapies, but ongoing monitoring requirements and adverse-event management are themselves potential equity barriers. Resmetirom requires baseline and periodic monitoring of liver biochemistries, lipid panel, thyroid function (TSH and free T4), and assessment for drug interactions; gastrointestinal adverse effects (diarrhea, nausea) are common in the first weeks of therapy and may require clinical contact for dose adjustment [6, 28]. Subcutaneous semaglutide requires patient education in self-injection, gradual dose titration to manage nausea and vomiting, and longitudinal contact for safety monitoring (including pancreatitis surveillance and assessment for gallbladder disease) [7, 8]. Each of these touchpoints presupposes reliable access to scheduled care, a phone or portal connection to the prescribing clinician, and the ability to leave work for appointments. Patients in safety-net systems and those facing transportation, language, or scheduling barriers are predictably more likely to miss monitoring visits, to discontinue therapy due to unaddressed side effects, or to fail to obtain dose adjustments. We therefore argue that equity-focused implementation of MASH pharmacotherapy must include (a) pharmacy-based or telehealth-delivered titration support, (b) translated dosing and side-effect material, (c) explicit accommodation for patients without reliable transportation, and (d) systematic measurement of disparities in persistence, not only in initiation.

3.9. Disparities in GLP-1 Receptor Agonist Prescribing

Emerging data reveal significant disparities in the prescribing of GLP-1 receptor agonists that may extend to their use in MASH. However, these data are derived from prescribing for obesity rather than for MASH itself. An analysis of semaglutide and tirzepatide prescribing for obesity in the U.S. from June 2021 through October 2024 demonstrated that while overall prescription rates increased substantially, non-Hispanic White patients maintained consistently higher prescription rates compared with Hispanic, non-Hispanic Asian, and non-Hispanic Black patients [31]. Patients in areas with the lowest social vulnerability showed more pronounced uptake than those in the most vulnerable areas. Metropolitan residents had higher prescription rates than rural populations [31]. These disparities persisted throughout the study period, including during drug-shortage intervals, suggesting that systemic barriers to access, rather than temporary supply constraints, drive inequitable uptake [31]. It bears repeating that these prescribing data reflect use for obesity (an indication with substantially different payer coverage policies, prior-authorization criteria, and out-of-pocket costs from MASH) and have been used here as an indirect proxy for likely patterns of access once semaglutide is prescribed for the F2 – F3 MASH indication. Direct measurement of MASH-specific prescribing disparities will not be possible until adequate post-approval claims data have accrued, and the projected disparities discussed in this section should therefore be interpreted as hypothesis-generating rather than as directly measured.

Medicare Part D formulary coverage for GLP-1 receptor agonists has expanded rapidly, with all three major agents (injectable semaglutide, oral semaglutide, and injectable tirzepatide) achieving over 90% plan coverage by 2024 [30]. However, this near-universal formulary adoption has been accompanied by a surge in prior-authorization requirements, suggesting that plans have shifted from restricting access through formulary exclusion to managing utilization through administrative barriers [30]. Medicare Advantage plans maintained persistently higher coverage rates than standalone prescription drug plans [30].

3.10. Underrepresentation in Clinical Trials: Beyond Enrollment Percentages

A systematic review and meta-analysis of 112 MASH randomized controlled trials encompassing 19,516 participants found that race and ethnicity were reported in only 61.6% and 50.0% of trials, respectively (Table 2) [32]. The pooled proportions of participants by race were: White 87.6%, Asian 5.0%, Black 2.3%, and Hispanic/Latino 31.4% (the last reported on the Hispanic/Latino ethnicity axis,

not as a race category) [32]. While female and Hispanic/Latino representation has increased over time, Black individuals remain severely underrepresented [32]. No reporting of sexual or gender minorities was identified [32].

The downstream consequences of this underrepresentation extend well beyond a generic concern about generalizability. We list the specific efficacy, safety, pharmacogenomic, and implementation questions that remain unanswered due to the demographic composition of the existing MASH evidence base. (1) Treatment-effect heterogeneity. MAESTRO-NASH enrolled approximately 20 Black participants; estimates of resmetirom efficacy in this group rest on extremely wide confidence intervals and are essentially uninformative for guideline-level inference. The ESSENCE interim publication did not separately report Black, Asian, or Hispanic/Latino response rates. (2) PNPLA3-stratified response. Tirzepatide pilot data suggest that PNPLA3 G allele carriers may have lower MASH resolution rates; resmetirom appears unaffected by genotype; PNPLA3 data for semaglutide are not yet published [12]. Because PNPLA3 allele frequency tracks with genetic ancestry, this raises a directly clinically important question of whether population subgroups with high G allele frequency will show systematically different response to GLP-1-based agents than to thyroid-receptor agonists. (3) Safety in comorbid populations. Real-world Black patients have higher rates of chronic kidney disease, atypical antipsychotic exposure, and hypertension medication exposure than the trial populations; these conditions interact with semaglutide dose escalation and with resmetirom's drug-drug profile. (4) Body-composition-specific dose-response. South Asian patients develop MASLD at lower BMI thresholds; whether weight-based dose titration of semaglutide is appropriate in this group is not addressed by current trials. (5) Adherence and persistence. Trial adherence is generally higher than real-world adherence; the gap is greater in groups facing more access and SDOH barriers, so trial-derived efficacy estimates will systematically overstate real-world effectiveness in underrepresented groups. (6) Hepatocellular carcinoma surveillance. No pivotal trial has powered an HCC endpoint, but Hispanic/Latino patients have higher MASLD-attributable HCC incidence; pharmacotherapy effects on HCC in this group are unstudied. Addressing these gaps will require post-approval observational studies and confirmatory trials with explicit enrollment goals, not merely descriptive reporting of demographics.

4. Discussion

4.1. Selection, Ascertainment, and Referral Bias

Most of the racial and ethnic differences described above are drawn from studies whose source populations sample fundamentally different patient pools. NHANES samples the U.S. general population using a probability design; the meta-analyses of Hispanic MASLD prevalence pool primarily community and primary-care cohorts; the NASH CRN biopsy cohort comprises patients who reached a tertiary academic center and underwent liver biopsy; the UNOS registry comprises patients who were referred, evaluated, and listed for liver transplantation; and pivotal pharmacotherapy trials comprise volunteers who passed multiple eligibility gates at specialized trial sites. Each filter is itself patterned by race, ethnicity, insurance, geography, and SDOH. Patients in underserved settings are less likely to be biopsied (so they appear with lower estimated severity in biopsy series), less likely to be listed for transplantation (so they appear less often in registry analyses), and less likely to enroll in trials (so they contribute little to efficacy estimates). The same individual may therefore appear across the studies cited in this review with an apparently lower risk of severe MASLD (because they never underwent biopsy), an apparently lower transplant probability (because they were referred late or not at all), and an apparently lower treatment response (because they were never enrolled). Some of what reads as racial or ethnic difference in the data therefore reflects how the data are collected, not how the disease behaves. Throughout the present review, we have tried to preserve this distinction; the findings should be read as the disparities produced by the existing care system, not as fixed biological characteristics of the groups being described.

4.2. Intersectionality Across the Care Continuum

Disparities in MASLD care do not affect every member of a racial or ethnic group identically. The transplant literature already documents intersectional effects of sex (non-White women with NASH cirrhosis are transplanted at higher MELD scores than White women and have worse post-transplant outcomes [27]). Still, the same multidimensional analysis should be applied across the care continuum. Age modifies FIB-4 performance and access to specialist care; rurality modifies access to VCTE and MRE, to hepatology, and to transplant centers; insurance type and immigration-related health-system engagement modify access to all of the above and to pharmacotherapy; language and health literacy modify both screening uptake and persistence on therapy; socioeconomic position interacts with food insecurity to modify even the protective effect of education [17]. For most of these axes, MASLD-specific intersectional data are scant: most cited studies stratify by either race/ethnicity OR sex OR insurance, but rarely by more than one simultaneously. Where such data exist (most prominently in the transplant literature), we have presented them; where they do not, we explicitly name the gap. Future research on disparity in MASLD should treat single-axis race/ethnicity reporting as a starting point rather than an endpoint.

4.3. An Equity-Focused Framework for U.S. MASLD Care

Recommendations in this section are stratified into three categories: (i) Current clinical actions are supported by current evidence and current guidelines and can be implemented now without further validation; (ii) Health-system implementation priorities are actions for health systems, payers, and policy makers that are reasonable given current evidence but require system-level investment to deliver; and (iii) Research gaps requiring validation are promising directions that should not be implemented in routine care until prospective evaluation is available.

4.3.1. Current Clinical Actions

Clinicians should: (1) universally apply FIB-4 screening for patients with type 2 diabetes, prediabetes with cardiometabolic risk factors, or obesity, regardless of race or ethnicity, per ADA and AACE guidance [4, 18, 19]; (2) when interpreting FIB-4 results, account for age and comorbidity rather than relying on a single fixed cutoff in all patients (see Differential Performance of Noninvasive Tests); (3) refer indeterminate or elevated FIB-4 results for VCTE or MRE per existing pathways and document and address barriers to imaging access; (4) screen patients with MASLD for food insecurity, transportation barriers, language preference, insurance coverage, and depression as part of routine assessment [23, 24]; (5) discuss the access pathway for resmetirom and semaglutide candidly with eligible patients, including prior authorization, copay, monitoring requirements, and pharmacy logistics; and (6) refer eligible patients to manufacturer patient-assistance and copay-support programs.

4.3.2. Health-System Implementation Priorities

Health systems and payers should: (1) invest in community-based VCTE capacity in safety-net systems serving high-prevalence populations rather than concentrating advanced imaging at tertiary

centers [24]; (2) integrate FIB-4 ordering into primary-care and endocrinology workflows with decision support that surfaces social-needs screening alongside the laboratory result; (3) reduce prior-authorization friction for FDA-approved MASH therapies, particularly in Medicaid and Medicare Advantage [30]; (4) expand pharmacy- and telehealth-delivered titration and adherence support for resmetirom and semaglutide, with translated materials; (5) develop transplant-center quality metrics that include race/ethnicity- and sex-stratified time-to-listing, time-to-transplant, and post-transplant survival [26, 27]; and (6) require structured measurement and public reporting of disparities in MASLD diagnosis, treatment initiation, treatment persistence, and outcomes.

4.3.3. Research Gaps Requiring Validation

The following are proposed for further investigation and are not recommended for routine clinical use until validated: (1) prospective validation of population-specific FIB-4, VCTE, and MRE thresholds in diverse, community-based U.S. cohorts with explicit subgroup reporting by Hispanic/Latino subgroup (Mexican American, Central/South American, Caribbean Hispanic) and Asian subgroup (East/South/Southeast Asian) [22]; (2) PNPLA3- and genetic-ancestry-stratified efficacy and safety analyses for resmetirom, semaglutide, and emerging agents in adequately powered trials or registries [12]; (3) equity-weighted cost-effectiveness analyses of MASH pharmacotherapy that incorporate downstream costs of untreated MASH in high-risk populations [29]; (4) mechanistic studies of the non-Hispanic Black biopsy-cohort paradox that control for ascertainment and referral effects [13]; (5) implementation-science evaluations of community-health-worker, pharmacist-led, and culturally tailored care models for MASLD [12]; and (6) intersectional disparity research that simultaneously stratifies by race/ethnicity, sex, age, rurality, insurance, and language. The FDA's 2024 final guidance on Diversity Action Plans provides a regulatory framework that supports the trial-side components of this agenda; sponsors are now expected to submit prospective enrollment goals stratified by race, ethnicity, sex, and age for most pivotal trials. We suggest, as expert opinion, that pre-specified subgroup analyses by race, ethnicity, and PNPLA3 genotype be planned for pivotal MASH trials and that trials be powered (or, when this is infeasible, descriptively reported) to permit meaningful interpretation of these subgroups; mandating such analyses for every pivotal trial would, however, be a policy choice for regulators rather than an evidence-based requirement.

5. Limitations

This review has several limitations. First, as a narrative, non-systematic review, it does not employ the methodology of a formal systematic review or meta-analysis, and selection bias in study inclusion is possible. Second, the MASLD nomenclature was adopted in 2023, and most cited studies used NAFLD/NASH criteria; concordance between the two definitions is high, but the introduction of the MetALD category may reclassify a subset of patients. Third, the evidence base for race/ethnicity-specific screening thresholds is derived from a single prospective study with external evaluation; broader prospective validation is needed. Fourth, data on disparities in access to newly approved MASH therapies are limited by the recency of FDA approvals, and our discussion of GLP-1 disparities is based on obesity prescribing as an indirect proxy. Fifth, the review is U.S.-focused, and findings are not generalizable to other countries or health systems. Sixth, most cited estimates derive from studies that did not adjust uniformly for the full set of relevant confounders, and we have therefore tempered causal interpretation throughout. Seventh, the populations grouped under "Hispanic/Latino" and "Asian" are highly heterogeneous; most underlying studies report only at the broad-group level, and this constrains our discussion.

5.1. Future Directions

Critical knowledge gaps warrant investigation, building on the research priorities listed above: (1) prospective validation of subgroup-specific noninvasive test cutpoints with explicit reporting at the level of Hispanic/Latino and Asian subgroups; (2) adequately powered clinical trials and post-approval observational studies of resmetirom, semaglutide, and emerging agents with pre-specified enrollment targets for Black, Hispanic/Latino, and Asian populations; (3) real-world effectiveness, persistence, and safety studies; (4) health-services research mapping insurance coverage and out-of-pocket costs by payer and by race/ethnicity, including the MASH-specific prior-authorization landscape once claims data accrue; (5) mechanistic and ascertainment-controlled studies of the non-Hispanic Black biopsy paradox; (6) implementation-science evaluation of culturally tailored screening programs, pharmacist-led titration, and community-health-worker interventions; and (7) investigation of the impact of the MASLD/MetALD nomenclature change on diagnostic patterns, referral rates, and patient engagement across racial/ethnic groups, including whether the removal of stigmatizing terminology improves care-seeking behavior.

6. Conclusion

MASLD care in the United States exhibits measurable disparities across multiple steps along the continuum from disease risk to advanced disease management, reflecting the interplay among genetic ancestry, social determinants of health, and structural barriers, rather than race or ethnicity as independent biological variables. Hispanic/Latino individuals have the highest reported MASLD prevalence among major U.S. racial and ethnic groups, a higher annual rate of prevalence increase than non-Hispanic Black and non-Hispanic White populations, reduced accuracy of standard non-invasive screening tools in the limited prospective data available, and lower observed transplant probability and higher waitlist mortality in registry analyses. Non-Hispanic Black individuals, while exhibiting lower MASLD prevalence on multivariable-adjusted population data, have higher mortality and lower transplant rates in studies of all-cause cirrhosis; MASH-specific outcome data in this population remain limited, and the contribution of biological factors versus care-access disparities cannot be separated with current evidence. The arrival of disease-modifying pharmacotherapies, resmetirom and semaglutide, represents a historic opportunity to alter the natural history of MASH, but, in light of cost, prior-authorization burden, monitoring demands, and the prescribing patterns observed for related agents in other indications, may widen existing disparities unless access is intentionally and equitably structured.

Achieving health equity in U.S. MASLD care will require coordinated action across multiple levels: validation and implementation of population-specific screening pathways; diversification of clinical trial populations with explicit attention to subgroup heterogeneity; reduction of financial, administrative, and monitoring-related barriers to treatment access; and investment in community-based interventions that address the social determinants driving disease burden. None of these proposed interventions has yet been demonstrated in MASLD-specific intervention studies to reduce racial or ethnic inequities; the case for them is based on observational disparity data, biological plausibility, and analogous evidence from related fields, and prospective evaluation is needed before any can be characterized as proven to narrow inequities. The renaming of NAFLD to MASLD offers a symbolic and practical opportunity to

reframe this disease in terms of its metabolic origins and to build U.S. care systems that serve all affected populations equitably.

Conflicts of Interest

The authors declare no relevant financial or non-financial conflicts of interest related to the subject matter of this manuscript.

Funding Source

This narrative review received no specific grant or other funding from any agency in the public, commercial, or not-for-profit sectors.

Acknowledgments

None.

Institutional Review Board (IRB)

Not applicable. This is a narrative review of previously published literature; no human subjects research was conducted.

Large Language Model

The authors used a generative artificial intelligence large language model (Anthropic Claude) to assist with copy-editing, language refinement, and editorial reorganization of a draft initially written by the authors, as well as reference formatting and cross-checking. The tool was not used to generate citations de novo, identify or select primary literature, draft scientific claims, or perform any independent literature search or analysis of the underlying study results. All references were subsequently verified individually, by DOI, against the primary sources by the authors. All clinical, epidemiologic, and policy content, all cited references, and all interpretations remain those of the authors, who have reviewed and verified the final manuscript and take full responsibility for its content. The nature and purpose of this tool use are described consistently in the manuscript, the cover letter, and the response to reviewers, and have been disclosed in accordance with ICMJE recommendations.

Author Contributions

RB contributed to conceptualization, methodology, investigation, writing the original draft, writing review and editing, visualization, and project administration. MK contributed to writing review and editing. ST contributed to investigation, writing review and editing, and supervision. All authors read and approved the final manuscript.

Data Availability

No original data were generated for this narrative review. All data discussed are available in the cited published literature. The complete final search strategy for each database is provided in the Supplementary Material (Appendix S1).

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